

Incidence of Hypertension in the Framingham Study

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Abstract: Incidence and trends in incidence of definite hypertension were analyzed based on 30 years follow-up of 5,209 subjects in the Framingham Heart Study cohort. Based on pooling of 15 two-year periods, hypertension incidence per biennium increased with age in men from 3.3 per cent at ages 30–39 to 6.2 per cent at ages 70–79, and in women from 1.5 per cent at ages 30–39 to 8.6 per cent at ages 70–79. No consistent trend in incidence rates was evident for

either sex from the 1950s through the 1970s. The proportion of hypertensive subjects receiving antihypertensive medication has increased since 1954–58 and exceeded 80 per cent for both men and women ages 60–89 years in 1979–81. Incidence data presented in this report may serve as a baseline for assessing the impact of future public health efforts in the primary prevention of hypertension. (*Am J Public Health* 1988; 78:676–679.)

Introduction

Few studies on incidence of hypertension in the general population have been published, although the prevalence of hypertension has been documented in a number of reports.^{1–3} No studies have reported trends in incidence in hypertension presumably due to the difficulty in collecting such data. Incidence of hypertension cannot be estimated from ambulatory care surveys because the vast majority of medical care for hypertension relates to prevalent rather than to incident cases. The availability of incidence data allows health care planners to focus primary prevention efforts as well as secondary prevention efforts in the highest risk groups. This report presents data on cumulative incidence and trends in incidence of hypertension based on three decades of follow-up of the Framingham cohort.

Methods

The Framingham Heart Study has followed a cohort of 5,209 men and women for more than 30 years with biennial examinations at the study clinic in Framingham, Massachusetts. Each examination included an extensive cardiovascular history and physical examination, an electrocardiogram, various blood chemistry determinations, and measurements of blood pressure, vital capacity, and other physiological variables. Morbidity and mortality were continuously monitored by hospital surveillance, contact with Vital Statistics for death certification, and communication with family physicians and relatives.

For exams prior to Exam 4 (1954–58), we assumed subjects were not taking antihypertensive medications based on the rarity of use of such medications in the early 1950s. Subjects were asked about use of antihypertensive medication beginning at Exam 4. In this report, use or nonuse of blood pressure medication was determined by the subject's response to this question for Exams 4 through 8. For Exams 9 (1964–68) through 16 (1979–81), subjects were considered to be taking antihypertensive medications only if, in addition to their report of use, the examiner indicated a clinical impression of "under treatment for hypertension."⁴ Use of medication in the interim period since the last exam was considered current use.

Systolic and diastolic blood pressure readings were taken from the left arm with a mercury sphygmomanometer while the subject was seated. Measurements were expressed in millimeters of mercury and were recorded to the nearest even number. During each examination, two blood pressure determinations were performed by a physician. Subjects were considered to have definite hypertension when both blood pressure readings were abnormal where abnormal was defined as systolic pressure of at least 160 mm Hg and/or diastolic pressure of at least 95 mm Hg. Subjects taking antihypertensive medications were also considered to have definite hypertension regardless of measured blood pressure level.

For each of 15 bienniums in the 30-year follow-up, subjects free of definite hypertension were followed for the development of definite hypertension to the next exam two years later. The observed number of subjects with new definite hypertension diagnosed at that next exam was divided by the number of individuals at risk at the beginning of the two-year period to obtain the two-year incidence rate. Observations from all 15 exams were pooled and age- and sex-specific two-year cumulative incidence rates were then calculated.⁵ At each exam, subjects were considered at risk of developing definite hypertension only if free of definite hypertension at all prior exams; presence or absence of other cardiovascular conditions were not considered in defining the population at risk.

To assess trends in incidence, the 30 years of follow-up were divided into three periods representing the 1950s (exams 2 through 6), 1960s (exam 7 through 11), and 1970s (exams 12 through 16). For each ten-year period, two-year age- and sex-specific incidence rates for that decade were calculated by pooling the observations from the five consecutive exams. Trends in incidence rates were examined by comparing the three successive decades.

Beginning with exam 4 (1954–58), the proportion of definite hypertensives on medication was calculated for every fourth biennial exam as the per cent of definite hypertensives who were receiving antihypertensive medication at that exam. All rates were calculated by sex and by age decade.

Results

The incidence of definite hypertension by age and sex based on pooling of 15 bienniums is presented in Figure 1. Incidence increased with age in men from 3.3 per cent at ages 30–39 to 6.2 per cent at ages 70–79, and in women from 1.5 per cent at ages 30–39 to 8.6 per cent at ages 70–79. The rate of increase with age was greater in women than men such that under age 50 men had a higher incidence of definite hyper-

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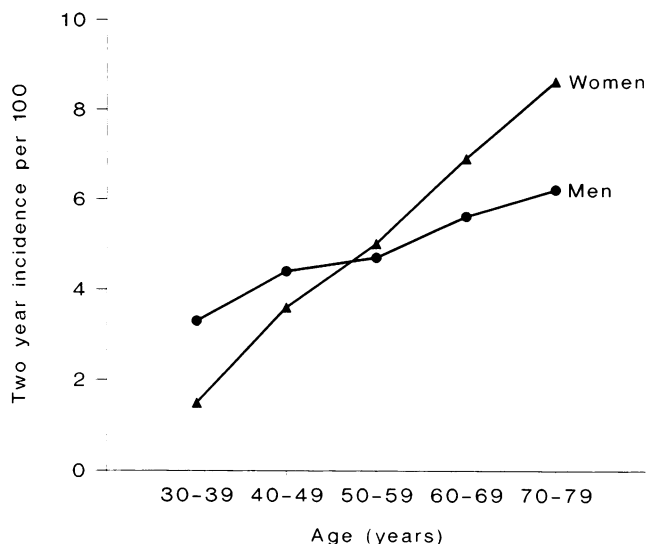


FIGURE 1—Two-Year Incidence of Definite Hypertension per 100 by Sex and Age Based on Pooling of 15 bienniums (Framingham cohort)

tension than women, while the opposite was observed among subjects older than age 50.

Changes in hypertension incidence over time were examined by comparing incidence rates for the 1950s, 1960s, and 1970s by sex and age (Figure 2). No clear trend in incidence rates is evident in any age group for either sex.

The proportion of hypertensive subjects receiving antihypertensive medication (Figure 3) increased consistently over time from 1954-58 to 1979-81 for both men and women in each age group studied. This proportion exceeded

80 per cent for both men and women ages 60-89 years in 1979-81. In cross-sectional analysis, the proportion receiving medication at each exam did not consistently vary with age.

Discussion

The increased incidence of hypertension with advancing age is consistent with previous reports⁶⁻¹² and may be due to factors such as decreased elasticity of the arterial wall¹³ and weight gain with age. While common in affluent societies, an increased incidence of hypertension with age is absent in some primitive cultures, apparently related to low sodium intake.¹⁴

Reasons for the higher incidence of hypertension in men than women under age 50, with a crossover above that age, are speculative. It is possible that men above age 50 at risk for hypertension are selectively removed from the at risk population by early death from cardiovascular disease. Alternatively, the poorly characterized genetic and environmental factors that cause hypertension may affect men at a younger age than women.

Most hypertension education programs have focused on the detection, treatment, and control of hypertension; considerable success has been achieved in those goals.³ The marked decline in cardiovascular disease in general and from stroke in particular over the past two decades¹⁵ is likely due in part to improved control of hypertension. However, substantially less attention has been given to the primary prevention of hypertension. Thus it is not unexpected that no clear downward trends in hypertension incidence were evident across the three time periods examined.

Whether modifying dietary sodium, obesity, and activity patterns in normotensive persons would substantially influence the incidence of hypertension is not known. This report focuses on the Framingham experience from 1948 to 1981;

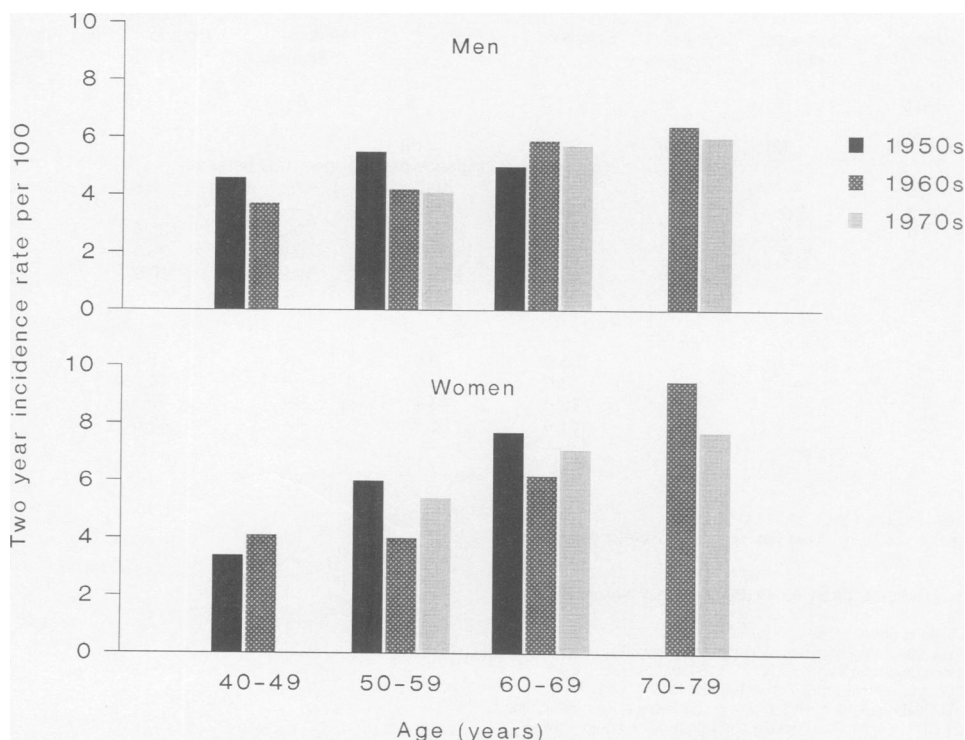


FIGURE 2—Trends in Two-Year Incidence of Definite Hypertension per 100 by Sex and Age Based on Examinations 2 through 6, 7 through 11, and 12 through 16 (Framingham cohort).

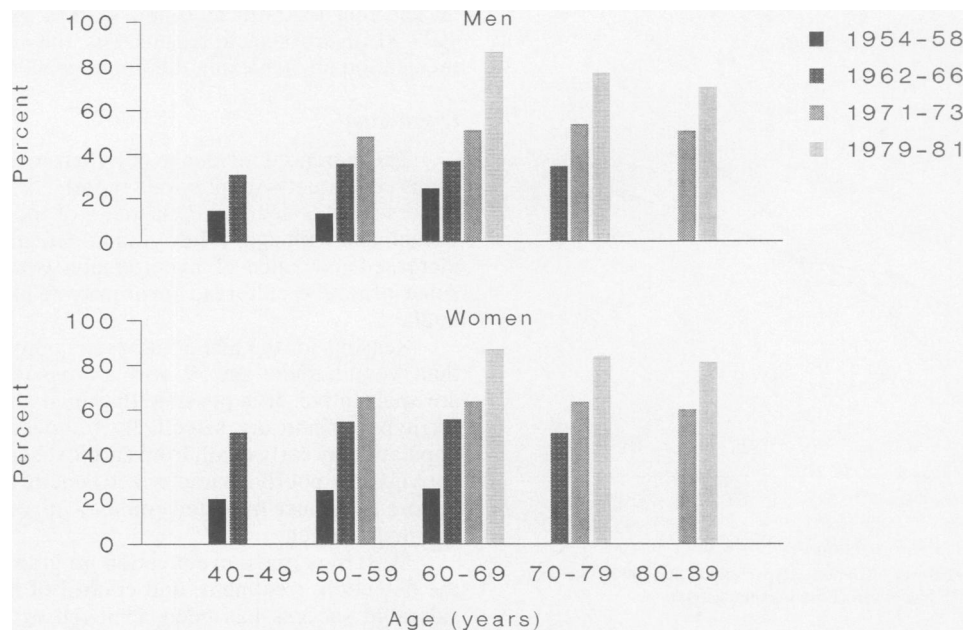


FIGURE 3—Per Cent of Subjects with Definite Hypertension taking Antihypertensive Medication by Sex and Age at Biennial Examinations 4, 8, 12 and 16 (Framingham cohort).

TABLE 1—Summary of Incidence of Hypertension in Selected Populations

Year	1944	1970	1972	1981	1982	1983	1987	1987	1988
Author	Levy ⁷	Dunn ⁸	Kahn ⁹	Dischinger ¹⁷	Andre ¹⁰	Paffenbarger ¹¹	Buck ¹²	Garrison ⁶	Present data
No. Men	22,741	11,614	3,829	1,092	5,373	14,998	4,559	1,418	2,236
No. Women	—	—	—	1,580	5,982	—	5,614	1,644	2,873
Study Period	1901-41	1950-64	1963-68	1973-77	1971-80	1962-72	1978-82	1971-83	1948-81
Age (years)	25-59	30-59	40-60+	30-69	20-60+	35-74	20-65	20-49	30-79
Race	not specified	Whites	mixed	Blacks	not specified	not specified	Whites predominantly	Whites	Whites
Location or Group	US Army officers	northeast US	Israeli civil servants	Baltimore	France	Harvard Alumni	Ontario	Framingham Offspring	Framingham Cohort
No. Years Follow-up	1-25+ ^a	5	5	3	5	6-10	5	8	30 ^b
Definition of Hypertension*	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(8)
Age Group (years)	Hypertension incidence per year per 1000 persons								
Men	20-29	5 ^c	—	—	10.0	—	6.8	5.5	—
	30-39	1.3	11.6	—	75.0	—	15.8	11.4	16.6
	40-49	3.9	23.0	7.8	71.0	5.5 ^e	25.8	17.8	21.9
	50-59	11.4	38.9	12.8	91.0	63.2	8.7 ^e	—	23.6
	60-69	—	—	20.4 ^d	62.3	88.6 ^d	10.2 ^e	40.8 ^f	28.0
	70-79	—	—	—	—	—	—	—	31.1
Women	20-29	—	—	—	2.4	—	4.6	2.0	—
	30-39	—	—	—	33.0	9.8	9.0	6.8	7.7
	40-49	—	—	—	57.0	25.0	20.8	16.1	18.0
	50-59	—	—	—	66.0	48.4	32.4	—	24.9
	60-69	—	—	—	61.0	95.8 ^d	42.6 ^f	—	34.7
	70-79	—	—	—	—	—	—	—	42.8

Notes:

- (a) 72% had 5 to 19 years of follow-up.
- (b) incidence data based on pooling of 15 two-year periods; see text for details.
- (c) ages 25 to 29 only.
- (d) ages 60 and older.
- (e) interpolated from data for 35-44, 45-54, 55-64 and 65-74 year age groups.
- (f) ages 60 to 65 only.

*Definitions of hypertension used in above studies.

- (1) at risk if no prior measured transient hypertension; hypertensive if SBP > 150 or DBP > 90.
- (2) a clinical diagnosis of hypertension and/or SBP >= 160 and/or DBP >= 95.
- (3) at risk if BP < 140/90; hypertensive if SBP >= 160 and DBP >= 95.
- (4) mean of 2nd and 3rd DBP readings >= 95 and/or on antihypertensive medication.
- (5) SBP >= 160 and/or DBP >= 95 and/or taking antihypertensive medications.
- (6) diagnosed by community physician.
- (7) DBP >= 90 at two consecutive visits to general practitioner's office.
- (8) both of two BP readings abnormal (where abnormal is defined as SBP >= 160 and/or DBP >= 95) and/or taking antihypertensive medications.

any public health efforts during the 1980s to lower hypertension incidence would be unlikely to have a detectable impact until the late 1980s or early 1990s.

Although the Framingham Study itself does not offer treatment, the increase in proportion of hypertensive subjects receiving medication could relate to secular changes in the prescription of antihypertensive medications by community physicians which may be different in other communities. However, the increase observed in Framingham is consistent with national trends.³

A summary of the incidence of hypertension in selected populations is presented in Table 1. In general the studies listed cannot be considered comparable because almost every study used a different definition of hypertension. The wide variation in incidence that may be expected when various definitions are used has been well documented.¹⁶ The definition used in the present report on the Framingham cohort was also used in the report on hypertension incidence in the Framingham offspring⁶; however, the age ranges of the subjects in the two studies only partially overlap. In addition, subjects in most prior studies were selected by employment,⁷⁻¹⁰ education,¹¹ race,¹⁷ or source of medical care,¹² and thus do not constitute representative samples of the general population.

Despite the lack of comparability, several observations about the studies listed in Table 1 are possible. Men had a higher incidence of hypertension than women in each age group in each study^{6,10,12,17,18} until approximately age 60, although a crossover appeared to occur somewhat earlier in the present study. Above age 60, the incidence of hypertension was at least as high or higher in women than men in the present study and in two other reports.^{10,17} Incidence of hypertension increased with age in eight out of nine studies; no clear age trend was evident in the study of Blacks in Baltimore¹⁷ nor in two other reports.^{16,19} The markedly higher incidence of hypertension in Blacks in the Baltimore study may be a function both of the definition of hypertension used and of the known higher incidence of hypertension in Blacks than in Whites.²⁰

Secondary prevention focused on hypertension diagnosis and treatment is the goal of current public health efforts. Although hypertension can be controlled by treatment, the high prevalence of antihypertensive medication use (especially among the elderly) and the adverse effects^{21,22} and costs of such drugs warrant concern. The eventual public health goal should be the primary prevention of hypertension and its sequelae. Evidence of successful primary prevention of hypertension may be reflected in incidence rates some years before being reflected in prevalence rates, due to increased diagnosis and improved survival among prevalent cases. This report presents incidence data that may be used to help target prevention efforts on the highest risk groups and to serve as

a baseline for assessing the impact of such future public health efforts in the primary prevention of hypertension.

REFERENCES

1. Folsom AR, Luepker RV, Gillum RF, *et al*: Improvement in hypertension detection and control from 1973-1974 to 1980-1981: The Minnesota Heart Survey experience. *JAMA* 1983; 250:916-921.
2. Freeman DH, Ostfeld AM, Hellenbrand K, Richards VA, Tracy R: Changes in the prevalence distribution of hypertension: Connecticut adults 1978-79 to 1982. *J Chronic Dis* 1985; 38(2):157-164.
3. Dannenberg AL, Drizd T, Horan MJ, Haynes SG, Leaverton PE: Progress in the battle against hypertension: Changes in blood pressure levels in the United States from 1960 to 1980. *Hypertension* 1987; 10:226-233.
4. Dannenberg AL, Kannel WB: Remission of hypertension: the "natural" history of blood pressure treatment in the Framingham Study. *JAMA* 1987; 257:1477-1483.
5. Cupples LA, D'Agostino RB, Anderson KM, Kannel WB: Comparison of baseline and repeated measure covariate techniques in the Framingham Heart Study. *Statistic Med* 1988; 7:205-218.
6. Garrison RJ, Kannel WB, Stokes J, Castelli WP: Incidence and precursors of hypertension in young adults: The Framingham Offspring Study. *Prev Med* 1987; 16:235-251.
7. Levy RL, Hillman CC, Stroud WD, White PD: Transient hypertension, its significance in terms of later development of sustained hypertension and cardiovascular-renal diseases. *JAMA* 1944; 126:829-833.
8. Dunn JP, Ipsen J, Elsom KO, Ohtani M: Risk factors in coronary artery disease, hypertension and diabetes. *Am J Med Sci* 1970; 259:309-322.
9. Kahn HA, Medalie JH, Neufeld HN, *et al*: The incidence of hypertension and associated factors: The Israel ischemic heart disease study. *Am Heart J* 1972; 84:171-182.
10. Andre JL, Monneau JP, Gueguen R, Deschamps JP: Five-year incidence of hypertension and its concomitants in a population of 11,355 adults unselected as to disease. *Eur Heart J* 1982; 3(Suppl C):53-58.
11. Paffenbarger RS, Wing AL, Hyde RT, Jung DL: Physical activity and incidence of hypertension in college alumni. *Am J Epidemiol* 1983; 117:245-257.
12. Buck CW, Donner AP: Factors affecting the incidence of hypertension. *Can Med Assoc J* 1987; 136:357-360.
13. Learoyd BM, Taylor MG: Alterations with age in the viscoelastic properties of human arterial walls. *Circ Res* 1966; 18:278-292.
14. Prior IAM, Evans JG, Harvey HPB, Davidson F, Lindsey M: Sodium intake and blood pressure in two Polynesian populations. *N Engl J Med* 1968; 279:515-520.
15. Kannel WB, Thom TJ: Declining cardiovascular mortality. *Circulation* 1984; 70:331-336.
16. Weissfeld JL, Kuller LH: Methodologic evaluation of incidence rates for hypertension: calculated for Pittsburgh's MRFIT usual care men. *J Chronic Dis* 1985; 11:915-925.
17. Dischinger PC, Apostolides AY, Entwisle G, Hebel JR: Hypertension incidence in an inner-city Black population. *J Chronic Dis* 1981; 34:405-413.
18. Schroll M, Hagerup L: Ten-year incidence of hypertension in the 1914-population in Glostrup. *Acta Med Scand* 1981; Suppl 646:15-18.
19. Robitaille NM, Dagenais GR, Rochon J, Lupien PJ: Incidence of arterial hypertension in the Quebec region. *Clin Invest Med* 1983; 6:39-42.
20. Apostolides AY, Cutter G, Daugherty SA, *et al*: Three-year incidence of hypertension in thirteen US communities. *Prev Med* 1982; 11:487-499.
21. Medical Research Council Working Party on Mild to Moderate Hypertension: Adverse reactions to bendrofluazide and propranolol for the treatment of mild hypertension. *Lancet* 1981; 2:539-543.
22. Multiple Risk Factor Intervention Trial Research Group: Multiple Risk Factor Intervention Trial: Risk factor changes and mortality results. *JAMA* 1982; 248:1465-1477.